

U.S. Department of Labor

Office of Administrative Law Judges
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Issue date: 15Aug2001

In the Matter of

Case No.: 2000-BLA-733

WALTER SEMSICK, JR.,

Claimant

v.

HELVETIA COAL COMPANY,

Employer

and

**DIRECTOR, OFFICE OF WORKERS'
COMPENSATION PROGRAMS,**

Party in Interest

Appearances:

Blair V. Pawlowski, Esq.
Pawlowski, Bilonick & Long
For the Claimant

George H. Thompson, Esq.
Thompson, Calkins & Sutter
For the Employer

Maria L. Spitz, Esq.
Office of the Solicitor
For the Director, OWCP

Before: Alice M. Craft
Administrative Law Judge

DECISION AND ORDER GRANTING BENEFITS

This proceeding arises from a claim for benefits under the Black Lung Benefits Act, 30 U.S.C. § 901 et seq. (the “Act”). The Act and implementing regulations, 20 CFR parts 410, 718, 725 and 727 (the “Regulations”), provide compensation and other benefits to: (1) living coal miners who are totally disabled due to pneumoconiosis and their dependents; (2) surviving dependents of coal miners whose death was due to pneumoconiosis; and (3) surviving dependents of coal miners who were totally disabled due to pneumoconiosis at the time of their death (for claims filed prior to January 1, 1982). The Act and Regulations define pneumoconiosis, commonly known as black lung disease, as a chronic dust disease of the lungs and its sequelae, including respiratory and pulmonary impairments, arising out of coal mine employment. 30 U.S.C. § 902(b); 20 CFR § 718.201 (2001). In this case, the Claimant, Walter Semsick, Jr., alleges that he is totally disabled by pneumoconiosis.

I conducted a hearing on this claim on October 4, 2000, in Pittsburgh, Pennsylvania. All parties were afforded a full opportunity to present evidence and argument, as provided in the Rules of Practice and Procedure, 29 CFR Part 18 (2001). At the hearing, Director’s Exhibits (“DX”) 1-31 (including exhibits 13A and 13B in addition to 13), Claimant’s Exhibits (“CX”) 1-11 and Employer’s Exhibits (“EX”) 1-9 were admitted into evidence without objection. Transcript (“Tr.”) 9-10 and 37. The record was held open after the hearing to allow the parties to submit additional evidence and argument. I hereby admit the following additional exhibits which have been submitted timely by the parties: Claimant’s Exhibit 12, the deposition of Dr. Schaaf; and Employer’s Exhibit 10, the deposition of Dr. Pickerill.

In reaching my decision, I have reviewed and considered the entire record, including all exhibits, the testimony at hearing and the arguments of the parties.

PROCEDURAL HISTORY

The Claimant filed his claim on August 9, 1999. DX 1. The Director of the Office of Workers’ Compensation Programs (the “Director,” “OWCP”) issued an Initial Determination granting benefits on March 8, 2000 (DX 24) and the Employer requested a formal hearing before the Office of Administrative Law Judges on March 23, 2000 (DX 27).

ISSUES

The issues contested by the Employer are:

1. How long the Claimant worked as a miner.
2. Whether the Claimant has pneumoconiosis as defined by the Act and the Regulations.
3. Whether the Claimant’s pneumoconiosis arose out of coal mine employment.

4. Whether the Claimant is totally disabled.
5. Whether the Claimant's disability is due to pneumoconiosis.

Other issues raised before the Director were waived, except for challenges to the regulations and the like which are not within my authority to consider. DX 30; Tr. at 5.

APPLICABLE STANDARD

This claim was filed after April 1, 1980. For this reason, the Regulations at 20 CFR Part 718 apply. 20 CFR § 718.2 (2001). In order to establish entitlement to benefits under Part 718, the Claimant must establish that he suffers from pneumoconiosis, that his pneumoconiosis arose out of his coal mine employment, and that his pneumoconiosis is totally disabling. 20 CFR §§ 718.1, 718.202, 718.203 and 718.204 (2001).

Parts 718 (standards for award of benefits) and 725 (procedures) of the Regulations have undergone extensive revisions effective January 19, 2001. 65 Fed. Reg. 79920 et seq. (2000). The Department of Labor has taken the position that as a general rule, the revisions to Part 718 should apply to pending cases because they do not announce new rules, but rather clarify or codify existing policy. See 65 Fed. Reg. at 79949-79950, 79955-79956 (2000). Changes in the standards for administration of clinical tests and examinations, however, would not apply to medical evidence developed before January 19, 2001. 20 CFR § 718.101(b) (2001). The new rules specifically provide that some revisions to Part 725 apply to pending cases, while others (including revisions to the rules regarding duplicate claims and modification) do not; for a list of the revised sections which do **not** apply to pending cases, see 20 CFR § 725.2(c) (2001).

On February 9, 2001, the United States District Court for the District of Columbia entered a *Preliminary Injunction Order* in a case challenging certain of the new rules, *National Mining Association, et al., v. Elaine L. Chao, et al.*, No. 1:00CV03086(EGS). Pursuant to ¶ 3 of the *Preliminary Injunction Order*, adjudication of claims pending before the Office of Administrative Law Judges on the effective date of the new regulations was stayed absent a finding, after briefing by the parties, that the new regulations would not affect the outcome of the case. On March 14, 2001, I issued an Order to Submit Briefs Addressing Whether Application of Amended Regulations Will Affect the Outcome of the Case. After receiving briefs from the Director and the Employer, on April 17, 2001, I issued an Order Finding Stay Inapplicable and Setting Time to Submit Closing Arguments. After I received closing argument from the Employer, while I was considering the case, on August 9, 2001, the District Court entered its decision upholding the new rules and dissolving the preliminary injunction. I will therefore apply the amended regulations where applicable. In this Decision and Order, the "old" rules applicable to this case will be cited to the 2000 edition of the Code of Federal Regulations; the "new" rules will be cited to the 2001 edition.

FINDINGS OF FACT AND CONCLUSIONS OF LAW

Factual Background and the Claimant's Testimony

The Claimant's last coal mine employment was in Pennsylvania. DX 2, DX 3. Therefore this claim is governed by the law of the Third Circuit. *Shupe v. Director, OWCP*, 12 B.L.R. 1-200, 1-202 (1989) (en banc).

The Claimant testified at the hearing of this matter. He was born on August 7, 1935. He is married to Alice Linda (Bauman) Semsick, who lives with him and is dependent on him for support. He admitted that his memory for dates was not very good, but said that this has been the case all of his life. He last worked in the mines in the early 1990's. He has been through several medical procedures since then, including surgery for his heart, removal of a leg, and removal of a lung because of lung cancer. He was using a wheelchair on the day of the hearing, but can walk with crutches. He recalled that all of his coal mining work was underground. His positions were buggy runner, utility man, miner operator, scoop runner, and braddish [phonetic spelling] man. The braddish man position involves laying block, and requires the miner to work on his knees and lift cement block over his head and carry sacks of cement. The Claimant felt this was a "hard job"; it was his last position. All underground jobs in the mines were very dusty. He submitted a photograph of himself and other miners leaving the mines on the way to the wash house. CX 11. He stated that he had breathing problems when he left the coal mines, and these problems got worse over time. Prior to losing his leg, the Claimant had to rest when doing activities. Tr. 11-18, 20-21, 37.

The Claimant recalled that he started smoking at age 15 or 16 and that he stopped smoking in 1995. He agreed that he had smoked for around 47 years. On days when he worked in the mines, he smoked a pack of cigarettes a day, and on days when he did not work in the mines, he smoked approximately a pack and a half a day. On cross-examination, the Employer noted that the Claimant had claimed in his July 16, 2000, statement that he began smoking in 1945, which would have been age 10. The Claimant admitted that he might have been about that age when he began to smoke. Tr. 18-20, 24.

The Claimant was cross-examined concerning written statements that his wife made on his behalf to the Employer dated July 16, 2000. The Claimant could not recall the date he left the mines, though the form indicated that it was on March 3, 1993. The Claimant had listed as his last position utility laborer, but he testified at the hearing that his last position involved building walls. He did not believe that the description of the physical demands of his position in the July 16, 2000 statement was accurate. He stated that when his wife completed the form, he gave the best answers he could. The Claimant could not recall the date he first had cardiac problems, but stated that he concurred with the medical records. He recalled having two heart operations. He also recalled having a portion of his lung removed for cancer, as well as having had cancer in the ear and surgery for an abdominal aneurism. He also had carotid artery surgery and amputation of his leg due to vascular disease. The Claimant has a

family history of heart disease and cancer. He stated that his breathing problems did exist prior to his heart problems, but that he did not run to the doctor for them, and only discussed them when he went to the doctor for things that had come up. The Employer called the Claimant's attention to Question Number 9 of his application for benefits, noting that the Claimant left blank this question, which dealt with disability due to pneumoconiosis. The Claimant stated that he filed for black lung benefits because Blue Cross told him that black lung should pay some of his bills. Tr. 20-35.

Length of Employment

The Employer agreed that the Claimant had 20 years and five months of coal mine employment. Tr. 5-6. The Claimant alleges that he had 27 years of coal mine employment (DX 30) but was willing to rest on the record before the court. Tr. 6. I have reviewed Helvetia Coal Company's statement regarding the Claimant's periods of employment in conjunction with the Claimant's Social Security records. DX 3, DX 4. They demonstrate that the Claimant was employed by Helvetia Coal Company from January 1971 to August 1994, which equals approximately 23 years and seven months. However, Helvetia Coal Company documented that the Claimant was out of work for several periods of time. A review of its statement shows that the Claimant missed approximately two years and five months of work. Accordingly, I find that the Claimant has established 21 years and 2 months of coal mine employment.

Medical Evidence

Chronological Discussion of the Claimant's Treatment for Cardiac and Pulmonary Conditions Beginning in 1992

In September of 1992, the Claimant was diagnosed with coronary artery disease, blood loss and anemia following surgery, and emphysema. Dr. David Evans examined the Claimant and rendered a report dated September 14, 1992. He noted that the Claimant had smoked 1 1/2 packs of cigarettes daily for the past 35 years. The Claimant denied cough, sputum production, hemoptysis, or orthopnea. Dr. Evans noted that the Claimant's lungs were clear to auscultation and percussion anteriorly, but that the chest was increased in the anteroposterior diameter. His impressions were "probable silent coronary artery disease," "probable chronic obstructive pulmonary disease," past basal cell carcinoma on the nose, and "probable peripheral vascular disease." Dr. Evans agreed that the Claimant should undergo cardiac catheterization, with possible "percutaneous transluminal coronary angioplasty." On September 1, 1995, the Claimant underwent a catheterization, which showed "mild left ventricular systolic dysfunction" and "severe three vessel coronary artery disease." Dr. Evans recommended that the Claimant undergo a coronary artery bypass grafting. Dr. Raj Devineni performed the coronary artery bypass surgery. During the Claimant's hospitalization, he underwent a number of chest x-rays, which are included in the x-ray chart. EX 2.

The Claimant underwent cardiac testing on March 27, 1995, including myocardial perfusion

imaging, a graded treadmill exercise test, and a doppler/echocardiogram. He underwent a chest x-ray on April 10, 1995, which showed a right upper lobe pulmonary mass, chronic obstructive lung disease, and post-surgical changes resulting from cardiovascular procedures. On April 11, 1995, the Claimant underwent lower arterial studies, which indicated "severe right superficial femoral artery occlusive disease and severe left aorto-iliac and/or superficial femoral artery occlusive disease." An upper arterial study done the same day showed "small vessel occlusive disease of the digits bilaterally." The Claimant was hospitalized after undergoing a cardiac catheterization by Dr. Evans on April 12, 1995. Based on the catheterization, Dr. Evans recommended that the Claimant consider a "rotational atherectomy of his left anterior descending coronary artery prior to pulmonary resection of the mass." However, Dr. Evans felt the Claimant could undergo diagnostic studies concerning the mass. EX 4.

On April 12, 1995, Dr. Johns and Dr. Leskovan (a resident) prepared a report summarizing the Claimant's examination and diagnosis. Examination of the chest showed "full respiratory excursion" and a symmetrical and grossly intact chest wall. The lungs were "clear to auscultation bilaterally" with well-nourished breath sounds and no rhonchi, rales or wheezing. Their impression was coronary artery disease, right lung mass, and currently-stable COPD. They recommended cardiac medication, and suggested that the Claimant undergo several diagnostic procedures and a pulmonary function test. EX 3.

Dr. Pickerill also examined the Claimant on April 12, 1995, and concluded that he had a "three centimeters right upper lobe apical segment lung mass" that was "very suspicious for bronchogenic carcinoma." He also diagnosed the Claimant with mild to moderate chronic obstructive pulmonary disease, bronchospasm, and coronary artery disease post surgery. He suggested that the Claimant undergo a bronchoscopy instead of a CT guided needle biopsy. He thought a lung resection would be likely, and recommended that a metastatic workup be conducted. He prescribed bronchodilators for the bronchospasm and chronic obstructive pulmonary disease. On April 13, 1995, Dr. Pickerill performed a "flexible bronchoscopy with transbronchial lung biopsies, apical segment right upper lobe, bronchial brushings and bronchial washings. Based on this procedure, he noted "no endobronchial obstructing lesions of proximal airways" and "no vocal cord paralysis." However, he found a "distal lung mass apical segment right upper lobe probably due to bronchogenic carcinoma." Dr. John Yerger, a pathologist, examined the lung tissue on April 13, 1995, and found "bronchogenic carcinoma, moderately differentiated squamous cell type." Based on the bronchial wash, he found "class V- cell block with squamous cell carcinoma." The bronchial brushings did not show squamous cell carcinoma cells. EX 3.

A whole body scan conducted on April 14, 1995, showed no osseous metastatic disease. However, it did show "two focal areas of focal soft tissue activity in the left neck and supraclavicular region." The Claimant's chest CT scan is summarized in the evidentiary charts. His abdominal CT taken on April 13, 1995, was unremarkable, except for an abdominal aortic aneurism. The Claimant's lung bases were clear. His head CT was unremarkable. EX 3. An April 14, 1995, letter from Dr. Martin B. Leon indicates that he reviewed the Claimant's coronary angiograms. The "catheter based options" for the Claimant included "treatment of the partially protected left main and LAD with a

combination of rotational atherectomy and stent implantation.” Dr. Leon felt it would be ideal to “perform the procedure without systemic anticoagulation.” He planned to transfer the patient and perform the procedure the following week. EX 5. The Claimant was transferred on April 16, 1995, to Washington Hospital Center for “PTCA of the mid left anterior descending artery lesion.” On April 17, 1995, the Claimant underwent a “successful PTCA of the mid left anterior descending coronary artery” performed by Dr. Leon. EX 4. Dr. Leon reported to Dr. Evans the details of the procedure and discussed the need to keep the Claimant on anticoagulation until his surgery for lung cancer. EX 5. After this procedure, once the Claimant was stable, he was to return to Conemaugh Hospital for surgery on his lung cancer. EX 3.

On April 22, 1995, Dr. Pickerill again consulted on the Claimant’s squamous cell carcinoma of the right upper lobe. Dr. Pickerill diagnosed the Claimant with a “three centimeter bronchogenic squamous cell carcinoma of the right upper lid apical segment,” “mild to moderate COPD” and coronary artery disease with past treatment. He recommended that the Claimant continue taking inhalers for his wheezing. He also recommended that the Claimant undergo additional pulmonary function studies before his surgery. He noted that a right upper lobectomy would probably be done. EX 1.

On April 24, 1995, Dr. Rajsekhar Devineni performed a mediastinoscopy, right thoracotomy, and right upper and middle lobectomy for removal of carcinoma of the right upper lung. In a May 2, 1995, letter to Dr. Johns, Dr. Devineni noted that the Claimant’s secondary diagnoses included chronic obstructive pulmonary disease, abdominal aortic aneurism, and coronary artery disease, post treatment. EX 1. Dr. David F. Stefanick treated the Claimant’s lung cancer with radiation. EX 1.

On May 13, 1996, the Claimant underwent a graded treadmill exercise test, which was negative. However, the Claimant “achieved only 62% of his predicted maximal HR which will lessen the sensitivity of the scan to depict any ischemia.” A myocardial perfusion imaging on the same day showed “no diagnostic evidence of exercise-induced reversible myocardial ischemia” though Dr. Oswald noted that the stress thallium myocardial perfusion scan was submaximal and “demonstrates no significant perfusion field defects.” There was “adequate thallium myocardial washout.” The “submaximal levels of stress lowers the sensitivity for detection [of] exercise-induced reversible myocardial ischemia.” EX 4.

One page of a Discharge Summary suggests that the Claimant underwent an “arch and bilateral carotid arteriogram” and “left carotid endarterectomy” in September of 1998. EX 6.

On June 1, 1999, the Claimant underwent a dobutamine stress EKG, which was “electrocardiographically equivocal for ischemia.” Palpitations were noted at peak stress with sinus tachycardia. A myocardial perfusion imaging conducted the same day was normal. EX 4.

Chest X-rays

Chest x-rays may reveal opacities in the lungs caused by pneumoconiosis and other diseases. Larger and more numerous opacities result in greater lung impairment. The quality standards for chest x-rays and their interpretations performed before January 19, 2001, are found at 20 CFR § 718.102 (2000) and Appendix A of Part 718. The following table summarizes the x-ray findings available in this case. Qualifications of physicians are abbreviated as follows: B= NIOSH certified B-reader; BCR= board-certified in radiology; BCP=board-certified in pulmonology; BCI= board-certified in internal medicine. Readers who are board-certified radiologists and/or B-readers are classified as the most qualified. See *Mullins Coal Co. v. Director, OWCP*, 484 U.S. 135, 145 n. 16 (1987); *Old Ben Coal Co. v. Battram*, 7 F.3d 1273, 1276 n.2 (7th Cir. 1993). B-readers need not be radiologists. Film quality codes are 1, Good; 2, Acceptable, with no technical defect likely to impair classification of the radiograph for pneumoconiosis; 3, Poor, with some technical defect but still acceptable for classification purposes; and 4 or U/R, Unacceptable. The existence of pneumoconiosis may be established by chest x-rays classified as category 1, 2, 3, A, B, or C according to ILO-U/C International Classification of Radiographs. A chest x-ray classified as category “0,” including subcategories 0/-, 0/0, 0/1, does not constitute evidence of pneumoconiosis. 20 CFR § 718.102(b) (2000). Small opacities (1, 2, or 3) (in ascending order of profusion) may be classified as round (p, q, r) or irregular (s, t, u), and may be evidence of “simple pneumoconiosis.” Large opacities may be classified as A, B or C, in ascending order of size, and may be evidence of “complicated pneumoconiosis.”

Exhibit Number	Date of X-ray/ Date Read	Reading Physician Name and Qualifications	Film Quality	ILO-U/C Class.	Interpretation or Impression
EX 2	09/15/92 09/16/92	McNiesh			No active disease.
EX 2	09/17/92 09/17/92	Mital			Satisfactory post-op[erative] cardiac surgery with no active chest disease.
EX 2	09/18/92 09/18/92	Abrahams			Stable examination from one day ago. The lungs are clear.
EX 2	09/19/92 09/20/92	Abrahams			Normal post-operative chest.

Exhibit Number	Date of X-ray/ Date Read	Reading Physician Name and Qualifications	Film Quality	ILO-U/C Class.	Interpretation or Impression
EX 2	09/23/92 09/23/92	Mital			Small amount of bilateral pleural effusion with postsurgical changes of cardiovascular procedure.
EX 3, EX 4	04/10/95 04/10/95	Mital			Pulmonary mass in the right upper lobe which was not present in 1992 and is highly indicative of malignancy...left lung field clear. Chronic obstructive pulmonary disease. Post-cardiovascular surgery changes
EX 3	04/13/95 04/13/95	Ringler			Satisfactory appearance to the chest post bronchoscopy.
DX 12	09/01/99 09/01/99	Khalaf, BCR	1		No pleural or parenchymal abnormalities consistent with pneumoconiosis; right upper lobectomy with marked right apical pleural thickening and some volume loss.
DX 13	09/01/99 09/11/99	Barrett, BCR, B	2		No pleural or parenchymal abnormalities consistent with pneumoconiosis (Other comments illegible)
CX 3	09/01/99 07/31/00	K.N. Mathur, BCR/B	1	1/1, p/s	Opacification of right apex is seen along with iatrogenic changes
CX 6	09/01/99 08/15/00	Brandon, BCR/B	2	2/2. q/t	Right apical capping, rule out cancer. Post op cxr. Status post CABG

Exhibit Number	Date of X-ray/ Date Read	Reading Physician Name and Qualifications	Film Quality	ILO-U/C Class.	Interpretation or Impression
EX 9	09/1/99 09/19/00	Palmer, BCR/B	1		No parenchymal or pleural abnormalities consistent with pneumoconiosis. Sutures in sternum and vascular clips in anterior mediastinum secondary to cardiac surgery. Vascular clips in right hilum which is retracted upward from volume loss, severe pleural capping and possibly surgery. Compensatory emphysema of right lower lung.
DX 19	11/18/99 11/18/99	Schaaf, BCP		1/0, p.	Right thoracotomy, median sternotomy, prior right upper lobectomy, pneumoconiosis
CX 1	11/18/99 07/13/00	K.N. Mathur, BCR/B	1	1/0, p/s	Right apex opacified, possibly fibrotic. Iatrogenic changes
CX 5	11/18/99 08/15/00	Brandon, BCR/B	2	2/2, q/t	Right apical capping, rule out cancer. Post op. Status post CABG
EX 9	11/18/99 09/18/00	Palmer, BCR/B	1		No parenchymal or pleural abnormalities consistent with pneumoconiosis. Previous cardiac surgery. Probable right thoracotomy with hilar retraction upward and severe pleural capping. Compensatory emphysema of right lower lung.”

Exhibit Number	Date of X-ray/ Date Read	Reading Physician Name and Qualifications	Film Quality	ILO-U/C Class.	Interpretation or Impression
DX 26	02/15/00 02/15/00	Pickerill, BCP/B	1	0/0	No parenchymal or pleural abnormalities consistent with pneumoconiosis. Right upper and middle lobectomies. Right apical pleural thickening. Previous CABG with wire [illegible word] the sternum and surgical clips in the mediastinum. DJD of thoracic spine. Flattening of diaphragm due to COPD. No change compared to x-rays of 5-7-98 and 9-2-98.
DX 26	02/15/00 02/21/00	Abrahams, B/BCR	1	0/0	No parenchymal or pleural abnormalities consistent with pneumoconiosis. Right upper lobectomy.
CX 4	02/15/00 07/31/00	K.N. Mathur, BCR/B	1	1/1, p/s	Opacification of right apex is seen along with iatrogenic changes
CX 7	02/15/00 08/15/00	Brandon, BCR/B	3	2/2, q/t	Right apical capping rule out cancer. Status post CABG. Post op.
EX 9	2/15/00 9/19/00	Palmer, BCR/B	2		no parenchymal or pleural abnormalities consistent with pneumoconiosis. "previous cardiac surgery." "Possible right thoracotomy with right hilum retracted upward and severe pleural capping" "compensatory emphysema of right lower lung field."

Biopsies

Biopsies may be the basis for a finding of the existence of pneumoconiosis. A finding of anthracotic pigmentation is not sufficient, by itself, to establish pneumoconiosis. 20 CFR § 718.202(a)(2) (2001). The quality standards for biopsies performed before January 19, 2001, are found at 20 CFR § 718.106 (2000). § 718.106(a) provides that a biopsy report shall include a detailed gross macroscopic and microscopic description of the lungs or visualized portion of a lung. If a surgical procedure was performed to obtain a portion of a lung, the evidence should include a copy of the surgical note and the pathology report. The Benefits Review Board has held, however, that the quality standards are not mandatory and failure to comply with the standards goes only to the reliability and weight of the evidence. *Dillon v. Peabody Coal Co.*, 11 B.L.R. 1-113, 1-114 (1988); *see Dagnan v. Black Diamond Coal Mining Co.*, 994 F.2d 1536, 1540-1541 (11th Cir. 1992). § 718.106(c) provides that “[a] negative biopsy is not conclusive evidence that the miner does not have pneumoconiosis. However, where positive findings are obtained on biopsy, the results will constitute evidence of the presence of pneumoconiosis.”

In this case, the biopsy report from the Claimant’s lung resection was not made a part of the record. Apparently, however, some of the reviewing physicians had access to this report, which included a gross and microscopic examination. According to Dr. Pickerill, who was a consulting and treating physician at the time of the Claimant’s diagnosis and treatment of the squamous cell carcinoma, the hospital pathologist found macular anthracosis and anthrosilicosis in the lymph nodes when conducting his examination for the purpose of detecting cancer. Dr. Perper, a pathologist who examined the microscopic slides himself, reported that the hospital pathologist, Dr. Yerger, made the following diagnoses:

- Bronchogenic carcinoma, squamous cell, moderately differentiated
- Proximal bronchial margin, free of carcinoma
- Fibrinous pleuritis with extension of carcinoma to beneath pleural surface
- Macular anthracosis
- Hilar lymph nodes: reactive hyperplasia with anthraco-silicosis.

Dr. Perper reviewed slides from the lung biopsy and resected right lung lobes, which consisted of “five glass slides, including the lung biopsy and four (4) slides from the resected right lung lobes.” Three of the slides contained a lung section; one contained five sections of lymph node. The lung biopsy contained three tiny fragments of tissue. On the biopsy, his diagnosis was squamous cell carcinoma of the lung, interstitial and solid pulmonary fibrosis, and pulmonary anthracotic pigmentation. He had noted anthracotic pigmentation scattered throughout the lung parenchyma, with occasional birefringent silica crystals and “small remnants of recognizable lung tissue with interstitial fibrosis.”

He reviewed the slides of the resected upper and middle lobes. He diagnosed moderately differentiated squamous cell carcinoma, simple coal workers’ pneumoconiosis of slight and primarily macular nature, moderately severe centrilobular emphysema, “interstitial and compact areas of

pulmonary fibrosis,” “sclerosis of small intra-pulmonary blood vessels consistent with pulmonary hypertension,” “chronic passive congestion” and “fibrinous pleuritis, focal.” He noted that “[t]he pleura shows moderate, focal, dense, fibro-anthracosis with presence of clusters of numerous birefringent silica crystals,” “marked solid and interstitial fibrosis,” “moderately severe centrilobular emphysema and areas of dense anthracosis with slight to moderate fibrosis, primarily around blood vessels and bronchioles.” He also noted “birefringent silica crystals in the anthracotic areas.” In the alveoli, he found many pigmented macrophages, which contained anthracotic pigment and “occasional birefringent silica crystals of fine granules of brown-yellow pigment consistent with hemosiderin.” The lymph nodes showed “focal anthracosis with presence of small numbers of birefringent silica crystals.” CX 9.

CT Scans

CT scans may be used to diagnose pneumoconiosis and other pulmonary diseases. The regulations provide no guidance for the evaluation of CT scans. They are not subject to the specific requirements for evaluation of x-rays, and must be weighed with other acceptable medical evidence. *Melnick v. Consolidation Coal Co.*, 16 B.L.R. 1-31, 1-33-1-34 (1991). EX 3 contains a report of a CT scans of Mr. Semsick’s chest taken on April 13, 1995.

Exhibit Number	Date of CT/ Date Read	Reading Physician	Interpretation or Impression
EX 3	4/13/95 4/13/95	Ringler	4 X 3 cm mass in right apex. Irregular margins. Abuts or extends into pleural surface. No evidence of obvious pulmonary nodules or hilar or mediastinal adenopathy.

Pulmonary Function Studies

Pulmonary function studies are tests performed to measure obstruction in the airways of the lungs and the degree of impairment of pulmonary function. The greater the resistance to the flow of air, the more severe the lung impairment. The studies range from simple tests of ventilation to very sophisticated examinations requiring complicated equipment. The most frequently performed tests measure forced vital capacity (FVC), forced expiratory volume in one-second (FEV₁) and maximum voluntary ventilation (MVV). The following chart summarizes the results of the pulmonary function studies available in this case. “Pre” and “post” refer to administration of bronchodilators. If only one figure appears, bronchodilators were not administered. The quality standards for pulmonary function studies performed before January 19, 2001, are found at 20 CFR § 718.103 (2000). The standards require that the studies be accompanied by two or three tracings of each test performed. In a “qualifying” pulmonary study, the FEV₁ must be equal to or less than the applicable values set forth in the tables in Appendix B of Part 718, and either the FVC or MVV must be equal to or less than the applicable table value, or the FEV₁/FVC ratio must be 55% or less.

Ex. No. Date Physician	Age Height	FEV ₁ Pre-/ Post	MVV Pre-/ Post	FVC Pre-/ Post	Compre- hension/ Cooper- ation	Qu al- ify	Physician Impression
EX 2 09/16/92 Kolff/ Devineni	57 67"	2.48 2.63		4.35 4.68	good effort and cooperation	no no	Moderate obstructive ventilatory defect. No significant improvement after bronchodilators. Lung volumes consistent with air trapping. Mildly reduced diffusing capacity.
DX 8 9/1/99 Bizousky	64 67"	1.78	65	3.29	good good	no	
DX 19 11/18/99 Schaaf	64 66" ¹	1.51 1.75	61 61	3.19 3.22	good effort	yes yes	Moderate obstructive lung disease. Significant improvement was noted post bronchodilators. Flow volume loop suggests no major airways obstruction.
DX 26 2/15/00 Pickerill	64 66"	1.78 1.93		3.32 3.26	good effort, cooperation and understan- ding	no no	Moderate obstruction, no restriction, no significant change post-bronchodilator. Lung volumes indicate hyperinflation. Moderately reduced DLCO. Flow volume loop consistent with obstruction.

¹The fact-finder must resolve conflicting heights of the miner recorded on the ventilatory study reports in the claim. *Protopappas v. Director, OWCP*, 6 B.L.R. 1-221, 1-223 (1983); *Toler v. Eastern Assoc. Coal Co.*, 43 F.3d 109, 114, 116 (4th Cir. 1995). As there is a variance of one inch in the recorded height of the miner, I must determine the miner's correct height. In this case, I accord more weight to the miner's height as recorded at the time of the pulmonary function test in 1992 (67"), before his left leg was amputated, because after the amputation it would be more difficult to obtain a correct height.

In his report and testimony, Dr. Pickerill refers to two pulmonary function tests from 1995. Like the biopsy reports, I have been unable to locate documentation of these tests in the evidentiary record. Therefore, I cannot accord much weight to these pulmonary function tests taken on March 22, 1995 and April 24, 1995. The values as described by Dr. Pickerill are not qualifying. They also have an FEV1/FVC ratio of between 54% and 59%.

Arterial Blood Gas Studies

Blood gas studies are performed to measure the ability of the lungs to oxygenate blood. A defect will manifest itself primarily as a fall in arterial oxygen tension either at rest or during exercise. A lower level of oxygen (O₂) compared to carbon dioxide (CO₂) in the blood indicates a deficiency in the transfer of gases through the alveoli which may leave the miner disabled. The quality standards for arterial blood gas studies performed before January 19, 2001, are found at 20 CFR § 718.105 (2000). The following chart summarizes the arterial blood gas studies available in this case. The blood sample is analyzed for the percentage of oxygen (PO₂) and the percentage of carbon dioxide (PCO₂) in the blood. A “qualifying” arterial gas study yields values which are equal to or less than the applicable values set forth in the tables in Appendix C of Part 718. If the results of a blood gas test at rest do not satisfy Appendix C, then an exercise blood gas test can be offered. Tests with only one figure represent studies at rest only. Exercise studies are not required if medically contraindicated. 20 CFR § 718.105(b) (2000). Exercise studies were not administered to the Claimant because of the loss of his leg.

Exhibit Number	Date	Physician	PCO ₂ at rest exercise	PO ₂ at rest exercise	Qualify	Physician Impression
DX 11	09/1/99	Bizousky	40	78	no	Slightly diminished pO ₂
DX 26	02/15/00	Pickerill	42	84	no	normal

Medical Opinions

Medical opinions are relevant to the issues of whether the miner has pneumoconiosis, whether the miner is totally disabled, and whether pneumoconiosis caused the miner’s disability. A determination of the existence of pneumoconiosis may be made if a physician, exercising sound medical judgment, notwithstanding a negative x-ray, finds that the miner suffers from pneumoconiosis as defined in § 718.201. 20 CFR §§ 718.202(a)(4) (2001). Thus, even if the x-ray evidence is negative, medical opinions may establish the existence of pneumoconiosis. *Taylor v. Director, OWCP*, 9 B.L.R. 1-22 (1986). The medical opinions must be reasoned and supported by objective medical evidence such as

blood gas studies, electrocardiograms, pulmonary function studies, physical performance tests, physical examination, and medical and work histories. 20 CFR § 718.202(a)(4) (2001). Where total disability cannot be established by pulmonary function tests, arterial blood gas studies, or cor pulmonale with right-sided heart failure, or where pulmonary function tests and/or blood gas studies are medically contraindicated, total disability may be nevertheless found, if a physician, exercising reasoned medical judgment, based on medically acceptable clinical and laboratory diagnostic techniques, concludes that a miner's respiratory or pulmonary condition prevents or prevented the miner from engaging in employment, i.e., performing his usual coal mine work or comparable and gainful work. 20 CFR § 718.204(b)(2)(iv) (2001). With certain specified exceptions, the cause or causes of total disability must be established by means of a physician's documented and reasoned report. 20 CFR § 718.204(c)(2) (2001). Quality standards for reports of physical examinations performed before January 19, 2001, are found at 20 CFR § 718.104 (2000). The record contains the following medical opinions relating to this case.

Dr. Bizousky

On September 1, 1999, Dr. F. P. Bizousky prepared an examination report concerning the Claimant. He noted the Claimant's history of heart disease, lung cancer, aneurism, left leg amputation, right ear cancer, and smoking history of one to one and one half packs of cigarettes per day from 1950 to 1995. The Claimant reported daily sputum production, some wheezing and coughing, and shortness of breath with limited activity. Dr. Bizousky noted that the Claimant had "multiple co-existing medical problems," which included severe peripheral vascular disease, coronary artery disease, left lung cancer with a partial pneumonectomy, and chronic obstructive pulmonary disease. The Claimant's chest x-ray indicated past coronary artery bypass graft surgery and pulmonary fibrosis. His pulmonary function studies indicated a combination of obstructive and restrictive pulmonary disease, and his arterial blood gas studies showed a slightly diminished pO₂. Dr. Bizousky diagnosed the Claimant with obstructive and restrictive pulmonary disease, based on his pulmonary function tests, his clinical history, the physical examination, and his coronary artery disease. He felt that the etiology of this impairment was smoking and mine dust exposure, as well as the Claimant's partial pneumonectomy. Dr. Bizousky characterized the Claimant's pulmonary impairment as severe, and did not feel that he could return to his last coal mine employment. He noted that the Claimant was impaired as a result of lung disease, heart disease, and severe peripheral vascular disease. The amputation was also a disabling condition, and resulted from the peripheral vascular disease. DX 10.

Dr. Schaaf

Dr. John T. Schaaf examined the Claimant and rendered a report dated November 22, 1999. DX 19. Dr. Schaaf is board certified in internal medicine, pulmonary disease, and critical care medicine. In his examination, Dr. Schaaf noted the Claimant's history of shortness of breath, daily sputum production and coughing when lying down. The Claimant did not wheeze. Dr. Schaaf noted a forty year smoking history of less than a pack a day. The Claimant reported 28 years of coal mine

employment. His positions were buggyman, miner helper, miner operator, running the scoop, and braddishman. His medical history included coronary artery bypass graft surgery, lung resection, abdominal aortic aneurism, ear surgery to remove cancer, and amputation following vascular procedures. Dr. Schaaf performed an x-ray and a pulmonary function test and reviewed the Claimant's medical records. His impression was that the Claimant had four problems. First, the Claimant had coal workers' pneumoconiosis, based on the x-ray and his mining history. Second, the Claimant had dyspnea brought on by "severe chronic airflow obstruction." This obstruction was the result of diminished lung volume after the resection of the Claimant's lung because of cancer and coal workers' pneumoconiosis. He felt that both of these conditions were "a significant contributing factor." However, since the Claimant was not in heart failure, it was unlikely that his dyspnea was significantly related to coronary artery disease. Third, the Claimant had a history of lung carcinoma, resulting in a bilobectomy. Fourth, the Claimant had coronary artery disease, and had undergone coronary artery bypass graft surgery.

Dr. Schaaf took an x-ray, which he felt clearly showed evidence of coal workers' pneumoconiosis, though at a low profusion. He noted that small nodules could not be seen on the right side because the Claimant's lobectomy distorted the parenchyma. The Claimant's pulmonary function test showed moderate obstructive lung disease, with significant improvement post-bronchodilator. The flow volume loop did not suggest major airways obstruction. DX 19.

Dr. Schaaf felt the Claimant's breathlessness was caused by severe obstructive airways disease, lung resection, and pneumoconiosis, and that his pneumoconiosis was a "substantial contributing factor to his breathlessness." He could not further quantify the amount of the contribution. He did not feel the Claimant could return to his last coal mine employment, due to multiple factors. DX 19.

Dr. Schaaf was deposed on October 16, 2000. CX 12. He reviewed his examination of the Claimant. He discussed the Claimant's symptoms and history. Dr. Schaaf concluded that the Claimant had sufficient dust exposure to be concerned about pneumoconiosis or other occupational lung diseases. He believed that coal workers' pneumoconiosis could progress after exposure to coal dust. Pulmonary function tests were performed in his office by a registered nurse, and the tracings and the Claimant's efforts were satisfactory and repeatable. The Claimant had a borderline significant improvement post-bronchodilator. Based on the pulmonary function test, Dr. Schaaf felt the Claimant had obstructive disease, though his vital capacity was at the lowest limit of normal. He noted that the Claimant's pulmonary function test taken in 1992 showed obstructive airways disease. He reviewed in more detail the Claimant's medical records beginning in 1995. These medical events post-dated the time period when the Claimant told him his shortness of breath began. Dr. Schaaf administered a chest x-ray, which he read as showing small round nodules predominantly on the left side. His impression was 1/0, p. He acknowledged on cross-examination that this is the earliest positive profusion and the smallest size opacity that can be categorized under the ILO classification system. Dr. Schaaf has never taken the test to become an A or B reader and has had no formal training under the ILO classification

system. However, he has had training in reading x-rays. Dr. Schaaf thought a reading of 0/1 could be accounted for by reader variation. He saw no evidence of complicated pneumoconiosis, progressive massive fibrosis or cor pulmonale. He further acknowledged that some medical literature suggests that a low profusion such as the Claimant's was not impairing. He noted that the Claimant's lung tissue had been found to show pneumoconiosis, and that examination of lung tissue is the gold standard for diagnosis of pneumoconiosis. Dr. Schaaf reviewed at least one set of cat scan films for the Claimant. The January 1999 cat scan was a standard technique film which Dr. Schaaf did not feel was appropriate for evaluating interstitial lung disease. This film was a 10 millimeter slice, which has reduced sensitivity for showing small nodules. A thinner slice would be needed to show nodulation or other interstitial changes. The 10 millimeter slice is appropriate for diagnosing malignancies.

Dr. Schaaf felt the Claimant's airflow obstruction was due to both his smoking and his coal workers' pneumoconiosis. He could not quantify the amount of contribution of each of these factors, but felt they were both significant contributing factors. Dr. Schaaf agreed that the Claimant had a sufficient smoking history to be considered an etiology for his impairment, but also that coal workers' pneumoconiosis can cause significant chronic obstructive pulmonary disease. Based on the Claimant's degree of impairment, Dr. Schaaf did not feel that the Claimant could return to his prior coal mining work. He acknowledged the variance between the smoking history he recorded, and the smoking history Dr. Pickerill recorded. He felt the Claimant had severe obstruction, based on the FEV1/FVC ratio of 47%. He noted that his pulmonary function test results were almost identical to those of Dr. Pickerill. Dr. Schaaf did not believe that a lung resection would significantly worsen obstruction, though it would cause a change in the total lung volume, which would manifest in the vital capacity. The other numbers would be proportionally reduced. The FEV-1/FVC ratio would not change, and that is the measure of obstruction. The Claimant's cardiac function was good based on a normal ejection fraction and could not account for his breathlessness or impaired exercise capacity. He also eliminated the Claimant's lung resection and squamous cell carcinoma as a cause of his shortness of breath because "most people who just have a lobectomy generally don't know it, they don't miss it." He felt the two possible causes of the Claimant's shortness of breath were smoking and coal workers' pneumoconiosis. He could not assign a percentage of causation to the two factors.

Ordinarily, Dr. Schaaf likes to see a positive x-ray to find pneumoconiosis. At the time he saw the Claimant, if he had found the x-ray negative, he would have attributed more of his pulmonary impairment to cigarette smoking. However, there was a record of the Claimant's lung resection in 1995, which, according to Dr. Schaaf, noted "multiple macular anthracosis measuring up to 0.3 centimeters in greatest dimension . . . [and] enlarged lymph nodes." Dr. Schaaf stated that the final diagnosis based on the lung resection was "macular anthracosis and then hilar lymph nodes, reactive hyperplasia with anthrosilicosis." The size of the densities in the pathology was only 3 millimeters, therefore it would not surprise Dr. Schaaf that these nodules could not be seen on x-ray. Dr. Schaaf believed that "the process leading to the nodules also leads to air flow obstruction and it causes distortion of lung architecture which sometimes leads to obstruction and can also lead to restriction." He felt it was a "complex process." The nodules themselves cause stiffness and a lack of elasticity,

which leads to obstruction and restriction. Dr. Schaaf acknowledged that the leading cause of pulmonary impairment in this country was cigarettes smoking. The smoking history he obtained was plus or minus one pack of cigarettes per day for approximately 40 years. Dr. Schaaf acknowledged that the Claimant had given a 96 pack year history of smoking when he was hospitalized for lung cancer in 1995. Since smoking can cause airflow obstruction, twice the smoking exposure would make the likelihood of this defect greater. Dr. Schaaf admitted that the Claimant had sufficient smoking history to account for the abnormalities on his pulmonary function studies. He has seen patients with the same level of impairment who have a smoking history of 40 plus years, but have never worked in the mines. If the Claimant did not have pneumoconiosis, Dr. Schaaf would attribute his impairment to his smoking disease.

Dr. Schaaf acknowledged that the Claimant's pulmonary function test showed borderline reversibility, and that pneumoconiosis is not a reversible disease. Officially, only asthma is reversible, but Dr. Schaaf has observed that "patients [with many diseases] get better when you give them bronchodilators." The Claimant did not have any restriction, and cigarette smoking does not cause restriction. He reviewed the numbers from the September 16, 1992, pulmonary function test and concluded that they showed moderate air flow obstruction. He may have referred to the FEV1 of 71 percent as mild. He would not rely on pulmonary function tests to determine whether a person could do a job; he did not ask the Claimant whether he felt he had the pulmonary capability to do his coal mine employment. He acknowledged that Dr. Chinsky had concluded that the 1999 pulmonary function test showed moderate obstruction, but he felt it showed severe obstruction, based on the fact that the FEV1 was 47 percent of his vital capacity. Dr. Schaaf thought most people would agree that this was severe. Based on Dr. Pickerill's chart of pulmonary function tests, the FEV1/FVC ratio was essentially the same before and after the surgical intervention. Dr. Schaaf felt that a person could be disabled from a pulmonary standpoint based on simple coal workers' pneumoconiosis, and he had determined someone in the past to be disabled when that person's x-ray reading was 1/0. There is not a direct correlation between profusion level and impairment level.

Dr. Schaaf did not believe that x-ray readings 1/0 and 2/2 were within the realm of reader variation; he did not see changes consistent with a 2/2 reading. He did not see Q or T type opacities. He did not see any changes on the right side of the lung, because the lung architecture was distorted as a result of the lobectomy. The lower lobe extended and became hyperinflated, decreasing his ability to see nodules and opacities. On examination, Dr. Schaaf did not hear any auscultation, percussion, wheezing, rales, or rhonchi. The Claimant had no clubbing or cyanosis. By physical examination there was nothing to indicate the Claimant had a pulmonary problem. In reaching his conclusions, Dr. Schaaf did not have any arterial blood gas studies, exercise testing, EKG's or cardiac testing to examine. Dr. Schaaf did feel the Claimant had normal heart function based on his 1995 ejection fraction of 40 percent; therefore, he did not feel that the Claimant's cardiac blockages could have caused shortness of breath on exertion. He did not retest the Claimant's ejection fraction in 1999 or 2000.

Dr. Schaaf felt that the Claimant's squamous cell lung cancer was caused by cigarette smoking,

but there was also an association between the cancer and pneumoconiosis. The link between pneumoconiosis and lung cancer was weaker than the link of smoking with lung cancer. Dr. Schaaf could not say that the Claimant's lung cancer was caused by coal dust exposure, but he could not exclude it as a potential cause of the cancer. The Claimant had severe vascular disease, but he did not believe it was a factor in the Claimant's shortness of breath.

Dr. Schaaf reviewed a New England Journal of Medicine, volume 243, pages 406-413 (February 10, 2000) article by Dr. William S. Beckett, which counsel for the Claimant provided to him. Dr. Schaaf agreed that this was a reputable journal and that the article suggested that coal and silica dust can cause chronic obstructive pulmonary disease and chronic airflow limitations, and that this disease can progress. However, cigarette smoking is the predominant cause of chronic obstructive pulmonary disease. Dr. Schaaf had not read the entire article and had not relied on it in treating or evaluating anyone. He was not aware of the nature of the supporting data for the article. He concurred with Dr. Beckett's finding that pneumoconiosis is progressive after exposure to coal dust and that coal workers' pneumoconiosis or coal dust exposure can cause obstructive airways disease. CX 12.

Dr. Pickerill

Dr. Pickerill examined the Claimant and rendered a report dated February 15, 2000. DX 26. He had previously consulted regarding the Claimant's lung cancer in 1995 and pulmonary atelectasis in 1996.² He reviewed the Claimant's occupational, smoking, family and medical histories, as well as his symptoms and medications. On examination, he heard no rales or wheezes. Pulmonary function tests showed a "moderate obstructive defect, but no restrictive defect." He did not find a significant change post bronchodilator. The Claimant's lung volumes were suggestive of hyperinflation, and his single breath carbon monoxide diffusion capacity was mildly decreased. His resting arterial blood gas study was normal, and an exercise test was not performed. The Claimant also underwent an EKG, which showed "minor ST-T wave flattening in the inferior leads, but no evidence of P-pulmonale." A chest x-ray was taken and read by Dr. Abrahams, who is a B reader and a board-certified radiologist (according to Dr. Pickerill), as 0/0. Dr. Pickerill, a B reader, also read the x-ray and interpreted it as 0/0. He noted "no significant change compared to the previous chest x-rays of 5-7-98 and 9-2-98." He did find COPD (emphysema), and a loss of volume in the right lung as a result of the lobectomies. Pleural thickening was found but was attributed to the Claimant's lung resection.

He reviewed the medical records of Dr. Schaaf and Dr. Johns, and summarized the x-ray readings by himself, Dr. Schaaf, and Dr. Abrahams. He found "no definite radiographic evidence of coal workers pneumoconiosis (category 0/0)." He did find evidence of COPD, lung resection, and coronary artery bypass surgery. He reviewed pulmonary function tests from 1992, 1995, 1999, and

²A summary of these evaluations is provided in the discussion of the treatment records and notes regarding the Claimant's treatment for cardiovascular and pulmonary problems.

2000, and concluded that they showed moderate COPD. He noted a 3% decrease in the pulmonary function studies after the April 24, 1995 right upper and middle lobectomies. The lung volumes indicated hyperinflation resulting from COPD, and the decreasing DLCO could "be attributed to the lung resection and COPD." Arterial blood gas studies from 1995 and 2000 were normal. DX 26.

Dr. Pickerill made several diagnoses. He found minimal coal workers' pneumoconiosis, based on the 1995 pathology findings. He also found moderate COPD and emphysema attributed to tobacco smoking, bronchogenic squamous cell carcinoma, coronary artery disease, squamous cell carcinoma of the right ear and right temporal bone, and left leg amputation due to ischemic peripheral vascular disease. Based on these diagnoses, Dr. Pickerill felt that the Claimant had minimal coal workers' pneumoconiosis and moderate chronic obstructive pulmonary disease due to smoking. "The minimal pneumoconiosis would only have a minor contribution to his moderate functional respiratory impairment, which [he] would attribute to COPD and the previous lung resection for bronchogenic carcinoma." The carcinoma was attributable to smoking, not coal mine employment. He did not think the Claimant's pneumoconiosis was "severe enough to cause a significant functional respiratory impairment and would not prevent him from doing his last job in coal mining industry from a respiratory standpoint." He felt the Claimant was disabled by many medical problems unrelated to coal mining, including COPD, lung resection, coronary artery disease, and left leg amputation. DX 26.

Dr. Pickerill was deposed on December 14, 2000. EX 10. He initially consulted on the Claimant's pulmonary condition in 1995. He performed a disability evaluation on February 15, 2000. He reviewed the Claimant's history. March 22, 1995, and April 24, 1995 pulmonary function tests showed moderate obstructive lung disease, "but it was similar to the pulmonary function studies done on 9-16-92 during my first consultation." The Claimant had wheezing and a prolonged expiration. He was given bronchodilators. In 1995, the Claimant had squamous cell carcinoma of the lung, associated with smoking. On April 12, 1995, Dr. Pickerill obtained the Claimant's smoking history, which was two packs of cigarettes per day for 40 years, ceasing in 1994. Dr. Pickerill knew of no "evidence that the squamous cell carcinoma would be due to coal mining exposure. The Claimant underwent a resection of the right upper lobe and right middle lobe, which represented a removal of two thirds of his right lung. The incidence of lung cancer in the general population of smokers compared to miner smokers, I don't think it's significantly different." The Claimant's coronary artery disease was not due to coal dust exposure. The Claimant's 1996 atelectasis was attributed to his aortic aneurism surgery.

At his 2000 examination of the Claimant, the Claimant reported a 23 year mining history, with his last job being brattice man. He obtained the same smoking history as in 1995. Dr. Pickerill recalled that shortness of breath was not a prominent complaint when the Claimant was still working. Rather, the complaint was recent and was the reason for the angina evaluation. The Claimant had had many other medical problems since Dr. Pickerill had last seen him. He performed a physical examination of the Claimant, and found no significant respiratory or cardiac problems other than the prior lung resection. The Claimant's x-ray was read by Dr. Abrahams, who is the chair of the Radiology Department at Memorial Medical Center. Dr. Abrahams did not find coal workers' pneumoconiosis,

though he did find evidence of chronic obstructive pulmonary disease. Dr. Pickerill also found chronic obstructive pulmonary disease, and read the film as 0/0. He interpreted the May 7, 1998 and September 2, 1998, films as 0/0 also. Based on the x-rays, he concluded that if the Claimant had coal workers' pneumoconiosis, it was of a "minimal, less than category one, type." The Claimant had undergone a biopsy and removal of lung with gross and microscopic examination. Dr. Pickerill interpreted those results as showing "only macular anthracosis changes, meaning there was dust pigment in the lung but it had not formed significant nodules for nodular pneumoconiosis."

Dr. Pickerill also performed a pulmonary function test, which showed a moderate obstructive defect, no restriction, hyperinflation based on lung volumes, and mild decreased carbon monoxide exchange. The arterial blood gas study was normal at rest, and an exercise study was not done. An electrocardiogram was not diagnostic. He reviewed other pulmonary function tests from 1992, 1995, 1999, and 2000. He stated:

All the pulmonary function studies showed evidence of moderate restrictive lung disease with a decrease in the FVC and FEV1. The results could primarily be attributed to the lobectomies of the right upper lobe and right middle lobe lobectomy. There was really no significant worsening of pulmonary functions from 1992 to 1995. They were similar to each study. These pulmonary functions showed no evidence of obstructive lung disease, hyperinflation of the lungs, which is typical for an obstructive type of lung disease rather than an interstitial fibrotic type of lung disease even in spite of the lobectomies.

Dr. Pickerill found that the Claimant had minimal coal workers' pneumoconiosis, based on the pathologist's finding of macular anthracosis and anthrosilicosis in the lymph nodes. Radiographically he found no coal workers' pneumoconiosis. He did not have an opportunity to review x-ray reports generated after his examination and report. Dr. Pickerill acknowledged that pathological examination was more sensitive than x-ray for "detecting lower grades of pneumoconiosis, in fact, even subclinical pneumoconiosis." Pneumoconiosis appears first in the upper lung zones. Dr. Pickerill relied on the pathologist's report, though he did review bronchoscopy biopsies and some of the slides following the resection. He did not review the gross examination of the lung. The absence of pneumoconiosis on bronchoscopy biopsy would not mean that it was not present. The bronchoscopy in this case was directed to the mass site. Dr. Pickerill had not reviewed Dr. Perper's report and review of the slides. Dr. Pickerill would not be in a position to decide from a pathology standpoint whether Dr. Perper of the hospital pathologist's findings were correct. Dr. Pickerill stated that it was not common practice to report minor degrees of emphysema found when doing pathology for lung cancer, and it would generally be reported only if it were extensive. With regard to whether the etiology of lung problems could be ascertained based on pathology, Dr. Pickerill stated:

I wouldn't say particularly. I would agree that if you did find extensive evidence of pneumoconiosis in the lung, that would be additional information. But pathology alone cannot ascribe the etiology of obstructive lung disease. In most cases, it's really the clinical

evaluations, physiological parameters, history that are the most reliable things.

If you find typical findings and there's differences, different types of so-called emphysema, like centrilobular emphysema, focal emphysema and these other factors that play into it. But that's additional information or data that's used for the overall assessment. It's not the exclusive data.

Dr. Pickerill acknowledged that pathology could be the gold standard in determining the type of emphysema present, but noted that "chronic obstructive lung disease is a conglomerate of diseases. It's not just emphysema. It's really a combination of chronic bronchitis, emphysema and other things." The hospital pathologist did not refer to either chronic bronchitis or emphysema in his report, though Dr. Pickerill did not believe that was unusual, since "they're not looking for those things" in a lung cancer case. The pathologist's choice to include findings of occupational lung disease was "just a personal preference." Dr. Pickerill agreed that it would be impossible to determine from that pathologist's report whether nodular disease, chronic bronchitis, or emphysema was present. He agreed that a pathologist who reviewed the slides specifically for occupational findings would probably be more reliable, except that he would only be able to do a microscopic examination, without a gross specimen. Hopefully, however, the samples would be representative, though he did not know whether they were. As a pulmonologist, Dr. Pickerill would look for nodules on a pathology report to diagnose coal workers' pneumoconiosis. He agreed that other descriptions such as silica crystals, birefringent crystals, and focal or centrilobular emphysema would be helpful. If another pathologist report found a greater extent of pneumoconiosis than found by the hospital pathologist, he would still rely on his clinical experience and testing rather than the pathology. On redirect, Dr. Pickerill was told that Dr. Perper had found "simple coal workers' pneumoconiosis, slight, primarily macular" and centrilobular emphysema. Dr. Pickerill felt centrilobular emphysema was "most commonly associated with cigarette smoking" and the pneumoconiosis finding was consistent with "mostly macular or pigment deposition and some other nodules presumably due to pneumoconiosis." He felt those findings were consistent with his opinions.

Dr. Pickerill's diagnosis of COPD was based on physiological findings, not pathological findings. The Claimant had moderate chronic obstructive pulmonary disease and emphysema, which could be attributed to smoking, and bronchogenic squamous cell carcinoma, also attributable to smoking. This type of lung cancer can occur in non-smokers, but is less likely to do so. He acknowledged that "fibrosis related to silica and other dust exposures can be related to lung cancer. It usually of the other types, generally not squamous cell carcinoma in my experience." It is more often an adenocarcinoma. He has seen a synergistic effect with silica exposure and cigarette smoking in causing lung cancer, but "more often when you can actually find fibrosis in the lung from the silica exposure." The Claimant had coronary disease, vascular disease, carotid stenosis, and squamous cell carcinoma of the ear, as well. He felt that the Claimant's minimal pneumoconiosis was not "severe enough to cause a significant functional respiratory impairment and would not prevent him from doing his last job in the coal mining industry from a respiratory standpoint." However, the Claimant could not do his last coal mine employment due to other medical problems.

Dr. Pickerill felt the Claimant's COPD and hyperinflation were attributable to smoking because they are "not typical for an interstitial fibrotic lung disease such as coal workers' pneumoconiosis." The Claimant had no restrictive defect prior to his lobectomy, and afterward had hyperinflation indicative of obstructive disease. This finding would be typical of pneumoconiosis only if it was complicated pneumoconiosis, of which there was no evidence. On cross examination, Dr. Pickerill agreed that simple coal workers' pneumoconiosis could cause chronic obstructive lung disease and diffusion capacity abnormalities. Prior to the lung resection, the Claimant would have had the respiratory ability to do his work, but not after the lung surgery. However, he acknowledged that the Claimant had a moderate obstructive defect before his surgery. His lung function decreased 30 percent after his surgery, though, indicating that the decrease was caused by the surgery, not worsening obstructive disease. After his resection surgery, the Claimant did not have a pulmonary function test until 1999. Dr. Pickerill acknowledged that coal workers' pneumoconiosis can progress after coal dust exposure, but "it usually applies when there is advanced pneumoconiosis. It's more likely to have progression without further exposure. . . . It's less likely when there is minimal and just minor categories of pneumoconiosis. It often does not progress significantly without further exposure." Dr. Pickerill felt it "would be very unlikely to have these degrees of decreased pulmonary function from pneumoconiosis alone without the lung resection with a minor degree of pneumoconiosis. It would be very unexpected." However, Dr. Pickerill acknowledged that "there could be a minimal contribution." Lobectomies cause a restrictive defect, not obstructive. He felt that the Claimant's minimal pneumoconiosis would only have a minimal contribution to his lung problems. "The majority or substantial lung problems were due to the chronic obstructive lung disease from smoking and also due to the resection of lung from the lung cancer." This determination was based on the "pulmonary function studies which were available before surgery and after surgery. The degree of involvement of the resected lung tissue by coal dust not showing significant nodular changes of pneumoconiosis, fibrotic changes of pneumoconiosis rather than just the macular dust accumulation." On cross-examination, Dr. Pickerill stated that pulmonary function tests do not "exclude or specifically eliminate any other factors." COPD caused by minimal pneumoconiosis does not usually cause severe or moderate obstructive defect, though COPD caused by "more advanced, obviously detected radiographic pneumoconiosis or complicated pneumoconiosis" might. Pathologically identified pneumoconiosis would have to be "fairly extensive" to cause such a defect. Category I pneumoconiosis can cause obstructive lung disease, but it is usually minimal or mild. Category II or III pneumoconiosis would produce more abnormalities on pulmonary function tests. Dr. Pickerill has seen patients with normal pulmonary function tests and complicated pneumoconiosis. He was aware of the conflict about the synergistic effect of smoking and dust exposure on obstructive lung disease, and he did "think that there is increased obstructive lung disease and coal dust exposure plus smoking."

With a left ejection fraction between 50 and 55 percent, coronary artery disease would not be expected to cause impairment at rest. However, a person "could get ischemia during exercise and have shortness of breath due to ischemia and left ventricular dysfunction could occur during exercise." The Claimant did not have a history of heart failure. The Claimant underwent a thallium study in 1995. This type of study is designed to show flow changes with exercise. On the Claimant's study, they did not

find reversible ischemia at 61% percent of the projected maximal stress. However, the submaximal level of stress made the test less sensitive to detecting myocardial ischemia. Ischemia could occur at a higher stress level. The goal is to achieve 85 percent of the maximum predicted. There is no further cardiac testing, though there were other problems. EX 10.

Dr. Perper

Dr. Joshua A. Perper prepared a report dated August 22, 2000, in which he reviewed records sent to him regarding the Claimant. CX 9. Dr. Perper is board-certified in anatomic pathology and forensic pathology. CX 10. He reviewed the Claimant's surgical pathology reports of April 14 and 24, 1995, in addition to other reports and records. He noted the smoking histories given to Dr. Schaaf and Dr. Pickerill, which were different from one another. He noted a 25 year mining history. He reviewed the Claimant's clinical history, including his 1992 hospitalization and coronary artery bypass surgery, and his 1995 cardiac catheterization and treatment for lung cancer. In particular, he summarized the pathological diagnoses of Dr. Yerger³ with regard to the lung specimen obtained during the surgery as follows:

- Bronchogenic carcinoma, squamous cell, moderately differentiated
- Proximal bronchial margin, free of carcinoma
- Fibrinous pleuritis with extension of carcinoma to beneath pleural surface
- Macular anthracosis
- Hilar lymph nodes: reactive hyperplasia with anthraco-silicosis.

He also noted the Claimant's 1996 surgery for an abdominal aortic aneurism, as well as his 1996 left femoro-popliteal arterial bypass graft, with subsequent complications ultimately resulting in a 1997 amputation of his leg. Also noted was his surgery for squamous cell carcinoma of the right ear canal extending into the right middle cranial fossa, as well as his 1998 carotid endarterectomy. Dr. Perper reviewed Dr. Bizousky's examination and report, as well as those of Dr. Schaaf and Dr. Pickerill. CX 9.

Dr. Perper reviewed five slides from the Claimant's lung biopsy and resected right lung lobes. His findings with regard to these slides are found in the discussion of biopsy evidence above. CX 9.

Dr. Perper determined that the Claimant had simple coal workers' pneumoconiosis based on the following factors: more than 25 years of exposure to coal mine dust; clinical symptoms such as cough, wheezing, mucus expectoration, obstructive impairment, and hypoxemia; some radiological

³Dr. Perper misspelled Dr. Yerger's name as "Yarger." Although the record does not contain Dr. Yerger's complete report, EX 3 contains a one-page Surgical Pathology Report from Dr. Yerger dated April 13, 1995, which confirms the correct spelling of his name.

findings of slight simple coal workers' pneumoconiosis; and pulmonary findings at autopsy [sic], which showed "mild simple coal workers' pneumoconiosis, primarily macular with associated interstitial and solid fibrosis, anthracotic pigmentation, silica crystals and centri-lobular (centri-acinar) emphysema." He noted that "centrilobular emphysema is a direct result of exposure to mixed coal mine containing silica and coal worker's pneumoconiosis." It is also a complication of heavy smoking. He further opined that the Claimant's coal workers' pneumoconiosis was due to his occupational exposure to coal mine dust, noting the presence of silica crystals in pneumoconiotic lesions in the Claimant's lungs, which shows exposure to coal dust containing silica. CX 9.

Dr. Perper noted that all reviewing and treating physicians had found the Claimant totally disabled due to many conditions and that he had simple coal workers' pneumoconiosis. He agreed with Dr. Schaaf that the Claimant's pneumoconiosis played a role in his total disability. Dr. Perper reviewed literature which suggests that emphysema can result from exposure to coal mine dust and that chronic obstructive pulmonary disease can result from a smoker's occupational exposure to dust. He noted studies that showed a correlation between emphysema in smokers and coal dust exposure. Dr. Perper opined that coal workers' pneumoconiosis was a substantial contributing cause of the Claimant's disability "both directly and through the associated centrilobular emphysema, that caused hypoxemia that either triggered or aggravated the myocardial ischemia associated with the arteriosclerotic heart disease or aggravated the patient's ischemic heart condition following the advent of the myocardial infarction." He also suggested that "a growing body of literature has substantiated a causal connection between exposure to mixed coal mine dust and coal workers' pneumoconiosis and the development of lung cancer." In particular, Dr. Perper noted that while lung cancer is related to heavy smoking, it has recently been related to occupational exposure to silica, which has been found to be carcinogenic in humans. Dr. Perper observed that many silica crystals were found in the Claimant's lung sections. Appended to Dr. Perper's report were two appendices, in which Dr. Perper reviewed literature discussing coal workers' pneumoconiosis as associated with centrilobular emphysema and coal workers' pneumoconiosis as associated with lung cancer. CX 9.

Dr. Branscomb

Dr. Ben V. Branscomb rendered a report dated August 8, 2000. EX 7. Dr. Branscomb is board certified in internal medicine and has an extensive background in pulmonary and respiratory medicine. He is a Distinguished Professor Emeritus of the University of Alabama at Birmingham. He reviewed the Claimant's medical records and the reports of Dr. Bizousky, Dr. Schaaf, and Dr. Pickerill. He concluded that the Claimant was suffering from "complications related to hardening of the arteries and also cancer including the lung." He noted that prior to these complications, the Claimant had not exhibited pulmonary symptoms, and that throughout the Claimant's treatment records, there was no reference to occupational pulmonary disease, except that of a physician in training and a medical student. He discussed the x-ray evidence, noting that the x-rays had "traced the development of the cancer and the distortions and scarring related to the resection." He felt that the "pleural thickening may well represent recurrent invasive cancer" and that "[t]he x-rays exclude visible CWP."

He reviewed the pulmonary function tests, and concluded that the September 16, 1992 pulmonary function study showed “mild airways obstruction with a slight degree of hyperinflation and air trapping.” He felt that these findings were typical for smokers, and that they might resolve if the Claimant stopped smoking. The September 1, 1992 [sic⁴] pulmonary function test conducted by Dr. Bizousky was invalid. Even if it were valid, it showed “a normal vital capacity and only moderate obstruction in spite of the removal of two fifths of the lung, subsequent distortion of the bronchi, and thickened pleura.” He felt Dr. Schaaf’s November 18, 1999, pulmonary function test and Dr. Pickerill’s February 15, 2000, pulmonary function test were “probably valid.” He concluded that the Claimant might have mild chronic obstructive disease. He noted that there was “no objective evidence of a disabling level of pulmonary impairment prior to the resection.” Prior to the surgery, the Claimant would have been able to engage in his coal mine work. EX 7.

Dr. Branscomb reviewed the arterial blood gas study from September 1, 1999, and concluded that the Claimant’s oxygen tension was not significantly low, and that the barometric pressure was extremely low. If one corrected for this, “the oxygen tension would have been 85.” He also reviewed Dr. Pickerill’s February 15, 2000, arterial blood gas study. Dr. Branscomb concluded that

there is no objective evidence that Mr. Semsick has any occupational pneumoconiosis, impairment or disability in any way caused or aggravated by either CWP or coal mine dust exposure. His lung cancer was neither caused nor aggravated by coal dust exposure. Indeed, some studies indicate lung cancer is slightly less common in miners than in the general population. The x-rays do not support the presence of pneumoconiosis nor do the pulmonary function studies.

He addressed Dr. Pickerill’s mention of the pathologist’s findings, but noted that they were “too indirect, lacking in detail, and confusing to allow me with reasonable confidence to conclude Mr. Semsick had CWP.” He defined anthracosis as carbon in the lung, and stated that a macule was a stain or spot. He felt that a macule was different from a coal macule. He felt that “antrhasilicosis of lymph nodes” was an “abandoned term.” EX 7.

Dr. Branscomb concluded that the Claimant probably was not disabled due to a pulmonary condition until after his cancer surgery. However, “he is totally disabled as a result of very severe vascular disease involving coronaries, aorta, legs, carotid artery, abdomen, and elsewhere. His disability is neither caused by nor aggravated by coal dust.” Even if the Claimant had “x-ray negative CWP” Dr. Branscomb would find that “it caused no impairment and aggravated no other condition.” He felt the Claimant had mild to moderate chronic obstructive pulmonary disease resulting from cigarette smoking, and not from dust. He noted: “Mild airways obstruction and bronchial

⁴Dr. Branscomb must be referring to the September 1, 1999, pulmonary function test conducted by Dr. Bizousky.

manifestations are sometimes associated with active coal mining. He was still mining in 1992 when the PFT's were obtained. Any dust effect of that type would have completely subsided within a few months of his retirement in 1994." EX 7.

Dr. Branscomb prepared a second report dated September 29, 2000. EX 8. He reviewed additional records, included Dr. Yerger's April 1995 report concerning the Claimant's lung specimen. Dr. Yerger found "multiple macular anthracosis," which Dr. Branscomb felt was consistent with smokers and miners. Dr. Yerger also found "anthraco-silicosis" in the hilar nodes, which Dr. Branscomb stated "generally means the finding of both carbon pigment and silica crystals were found in the lymph nodes which drain the lung." Dr. Branscomb felt this was a typical finding for miners and "does not constitute a disease in the lymph nodes." Additional records were reviewed. With regard to Dr. Bizousky's September 1, 1999, report, Dr. Branscomb noted that Dr. Bizousky did not have valid pulmonary function tests or access to the pre-surgery records. With regard to Dr. Schaaf's November 18, 1999, report, Dr. Branscomb "respectfully note[d] that there was no objective valid measurement of shortness of breath sufficient to prevent Mr. Semsick from returning to work." Dr. Branscomb felt the Claimant's cardiovascular disease explained his shortness of breath. He observed that Dr. Schaaf's x-ray reading was not consistent with the majority of readers. EX 8.

Dr. Branscomb also discussed Dr. Perper's report. He noted that Dr. Perper had found simple coal workers' pneumoconiosis of a slight and primarily macular nature and centrilobular emphysema. Dr. Branscomb noted that "the extent of identifiable emphysema on a microscope slide has no correlation whatsoever with the level of overall pulmonary function of the patient." He questioned Dr. Perper's notation that the Claimant had cough, wheezing, etc., because the Claimant did not have these problems prior to his "numerous non-occupational insults to the lung." Dr. Branscomb concluded that

I have no doubt that pulmonary conditions contribute to Mr. Semsick's current total disability, as Dr. Perper notes in his question 3 on page thirteen. However, based on the minuscule evidence of CWP microscopically plus the gravity of the massive lung resection, radiation therapy, shift of tissues from surgery, severe and multiple vascular problems certainly explain any pulmonary contribution. There is no particular reason either from the medical literature, Mr. Semsick's history, or the pathologic examination to think that the minimal microscopic CWP present contributed to any impairment much less to a total impairment.

He disputed Dr. Perper's conclusions and review of the literature regarding the relationship between coal workers' pneumoconiosis and emphysema, though he did not state with specificity what about them he disagreed with. He found no evidence in the record that the Claimant had "severe pulmonary emphysema of any etiology." Also, there was no evidence that the Claimant had any "disabling pulmonary impairment" before the series of catastrophic events from which he was fortunate even to have survived." He did not believe the pathology showed "sufficient CWP to expect dysfunction." However, based on the reports of Dr. Perper and Dr. Pickerill, Dr. Branscomb acknowledged that there "may well have been a minimal degree of microscopic CWP" but that "it had no adverse

functional effect whatsoever and clinically neither caused nor aggravated any other condition or impairment.” EX 8.

Existence of Pneumoconiosis

The regulations define pneumoconiosis broadly:

(a) For the purpose of the Act, “pneumoconiosis” means a chronic dust disease of the lung and its sequelae, including respiratory and pulmonary impairments, arising out of coal mine employment. This definition includes both medical, or “clinical”, pneumoconiosis and statutory, or “legal”, pneumoconiosis.

(1) *Clinical Pneumoconiosis*. “Clinical pneumoconiosis” consists of those diseases recognized by the medical community as pneumoconioses, *i.e.*, the conditions characterized by permanent deposition of substantial amounts of particulate matter in the lungs and the fibrotic reaction of the lung tissue to that deposition caused by dust exposure in coal mine employment. This definition includes, but is not limited to, coal workers’ pneumoconiosis, anthracosilicosis, anthracosis, anthrosilicosis, massive pulmonary fibrosis, silicosis or silico-tuberculosis, arising out of coal mine employment.

(2) *Legal Pneumoconiosis*. “Legal pneumoconiosis” includes any chronic lung disease or impairment and its sequelae arising out of coal mine employment. This definition includes, but is not limited to any chronic restrictive or obstructive pulmonary disease arising out of coal mine employment.

(b) For purposes of this section, a disease “arising out of coal mine employment” includes any chronic pulmonary disease or respiratory or pulmonary impairment significantly related to, or substantially aggravated by, dust exposure in coal mine employment.

(c) For purposes of this definition, “pneumoconiosis” is recognized as a latent and progressive disease which may first become detectable only after the cessation of coal mine dust exposure.

20 CFR § 718.201 (2001). In this case, Mr. Semsick’s medical records indicate that he has been diagnosed with chronic obstructive pulmonary disease and emphysema, which can be encompassed within the definition of legal pneumoconiosis. *Ibid.*; *Richardson v. Director, OWCP*, 94 F.3d 164 (4th Cir. 1996); *Warth v. Southern Ohio Coal Co.*, 60 F.3d 173 (4th Cir. 1995).

20 CFR § 718.202(a) (2001), provides that a finding of the existence of pneumoconiosis may be based on (1) chest x-ray, (2) biopsy or autopsy, (3) application of the presumptions described in §§ 718.304 (irrebuttable presumption of total disability if there is a showing of complicated

pneumoconiosis), 718.305 (not applicable to claims filed after January 1, 1982) or 718.306 (applicable only to deceased miners), or (4) a physician exercising sound medical judgment based on objective medical evidence and supported by a reasoned medical opinion. None of the presumptions apply, because the evidence does not establish the existence of complicated pneumoconiosis, the Claimant filed his claim after January 1, 1982, and he is still living. In order to determine whether the evidence establishes the existence of pneumoconiosis, therefore, I must consider the chest x-rays, biopsies and medical opinions. Absent contrary evidence, evidence relevant to any category may establish the existence of pneumoconiosis. In the face of conflicting evidence, however, I must weigh all of the evidence together in reaching my finding whether the Claimant has established that he has pneumoconiosis. *Island Creek Coal Co. v. Compton*, 211 F.3d 203, 211 (4th Cir. 2000); *Penn Allegheny Coal Co. v. Williams*, 114 F.3d 22 (3rd Cir. 1997).

Pneumoconiosis is a progressive and irreversible disease. *Labelle Processing Co. v. Swarrow*, 72 F.3d 308, 314-315 (3rd Cir. 1995); *Lane Hollow Coal Co. v. Director, OWCP*, 137 F.3d 799, 803 (4th Cir. 1998); *Woodward v. Director, OWCP*, 991 F.2d 314, 320 (6th Cir. 1993). As a general rule, therefore, more weight is given to the most recent evidence. *See Mullins Coal Co. of Virginia v. Director, OWCP*, 484 U.S. 135, 151-152 (1987); *Eastern Associated Coal Corp. v. Director, OWCP*, 220 F.3d 250, 258-259 (4th Cir. 2000); *Crace v. Kentland-Elkhorn Coal Corp.*, 109 F.3d 1163, 1167 (6th Cir. 1997); *Rochester & Pittsburgh Coal Co. v. Krecota*, 868 F.2d 600, 602 (3rd Cir. 1989); *Stanford v. Director, OWCP*, 7 B.L.R. 1-541, 1-543 (1984); *Tokarcik v. Consolidated Coal Co.*, 6 B.L.R. 1-666, 1-668 (1983); *Call v. Director, OWCP*, 2 B.L.R. 1-146, 1-148-1-149 (1979). This rule is not to be mechanically applied to require that later evidence be accepted over earlier evidence. *Woodward*, above at 319-320; *Adkins v. Director, OWCP*, 958 F.2d 49 (4th Cir. 1992); *Burns v. Director, OWCP*, 7 B.L.R. 1-597, 1-600 (1984).

Review of the record discloses that there are as many as twelve x-rays referenced in the record. There are radiological reports for seven x-rays taken during the Claimant's hospitalizations in 1992 and 1995. None of those radiological interpretations are classified for or mention coal workers' pneumoconiosis. In his deposition, Dr. Pickerill indicated he had reviewed x-rays from May 7, 1998, and September 1, 1998, and found them both to be 0/0. EX 10. There are three x-rays of record that have been read by multiple readers for the purposes of black lung disability evaluation. These three x-rays are also the most recently taken x-rays. Accordingly, I will give considerable weight to the findings regarding these three x-rays. Because all three were taken within five and one-half months of each other, between September 1, 1999, and February 15, 2000, however, the "later evidence" rule cannot be applied to distinguish among the three. *See Stanley v. Director, OWCP*, 7 B.L.R. 1-386, 1-388 (1984). Each has been read by some but not all reviewers to be positive for pneumoconiosis. For cases with conflicting x-ray evidence, the Regulations specifically provide,

Where two or more X-ray reports are in conflict, in evaluating such X-ray reports consideration shall be given to the radiological qualifications of the physicians interpreting such X-rays.

20 CFR § 718.202(a)(1) (2001); *Dixon v. North Camp Coal Co.*, 8 B.L.R. 1-344 (1985); *Melnick v. Consolidation Coal Co.*, 16 B.L.R. 1-31, 1-37 (1991). Readers who are board-certified radiologists and/or B-readers are classified as the most qualified. The qualifications of a certified radiologist are at least comparable to if not superior to a physician certified as a B-reader. *Roberts v. Bethlehem Mines Corp.*, 8 B.L.R. 1-211, 1-213 n.5 (1985). Greater weight may be accorded to x-ray interpretations of dually qualified physicians. *Sheckler v. Clinchfield Coal Co.*, 7 B.L.R. 1-128, 1-131 (1984). A judge may consider the number of interpretations on each side of the issue, but not to the exclusion of a qualitative evaluation of the x-rays and their readers. *Woodward*, 991 F.2d at 321; *see Adkins*, 958 F.2d at 52.

The x-ray taken on September 1, 1999, was read by five physicians. The interpretations of this x-ray are quite varied. Dr. Brandon, who is both a board-certified radiologist and a B reader, found a 2/2 profusion. Dr. Mathur, also dually qualified, found a 1/1 profusion. However, Dr. Barnett and Dr. Palmer, also dually qualified, found no evidence of pneumoconiosis, as did Dr. Khalaf, a board-certified radiologist. Given the wide variance in the readings, across equally qualified readers, and the fact that the majority of the readers found no pneumoconiosis, I find inadequate evidence to establish the existence of pneumoconiosis based on this x-ray.

Turning next to the x-ray of November 18, 1999, I note that there are four readings of this x-ray. Dr. Brandon, who is dually qualified, found a 2/2 profusion; Dr. Mathur, who is dually qualified, found a 1/0 profusion. Dr. Schaaf, who is neither a radiologist nor a B reader, found a profusion of 1/0 as well. In contrast, Dr. Palmer, who is dually qualified, found no evidence of pneumoconiosis on this x-ray. While Dr. Schaaf is certainly well qualified in the area of pulmonology, I accord his opinion on this x-ray less weight, based on the superior qualifications of other readers. Giving less weight to his opinion, I am still left with three divergent interpretations of the x-ray. All of the readers are dually qualified. Two of them found the existence of pneumoconiosis on the x-ray; one found no pneumoconiosis. Under the circumstances, I conclude that the November 18, 1999, x-ray is the strongest of the three indicative of coal workers' pneumoconiosis. Nonetheless, I do not find it conclusive.

I next consider the February 15, 2000, x-ray. There are five readings of this x-ray. Dr. Brandon, who is dually qualified, found a 2/2 profusion; Dr. Mathur, who is also dually qualified, found a 1/1 profusion. In contrast, Dr. Palmer (dually qualified), Dr. Pickerill (a B-reader) and Dr. Abrahams (a B reader and Board Certified Radiologist) found no pneumoconiosis. Once again, the readings are widely divergent, even among equally qualified readers. At best, the readings are in equipoise.

As the burden of demonstrating pneumoconiosis remains with the Claimant, I cannot conclude that three most recent x-rays, singly or in combination, establish the existence of pneumoconiosis.

I must next consider the biopsy evidence in this case. The report of the actual gross and microscopic examination performed by Dr. Yerger in 1995 inexplicably was not included in the exhibits

presented to me by the parties. However, Dr. Yerger's findings were summarized by various physicians who reviewed the records in this matter. While my inability to observe whether of the quality standards have been met makes an analysis of this evidence somewhat challenging, I note that many of the reviewing physicians reviewed the report and did not dispute the findings. Indeed, any dispute arising out of the report was limited solely to the issue of whether the findings were sufficient to make a finding of pneumoconiosis. According to Dr. Schaaf, the hospital pathologist's findings were multiple macular anthracosis measuring up to 0.3 centimeters in greatest dimension . . . [and] enlarged lymph nodes." The lung resection was indicative of "macular anthracosis and then hilar lymph nodes, reactive hyperplasia with anthrosilicosis." The size of the densities in the pathology was only 3 millimeters. In this case, I cannot accord weight to the opinion of the hospital pathologist, Dr. Yerger. While I do not doubt his qualifications, his report is not a part of the record. Furthermore, Dr. Pickerill opined that many pathologists who are performing examinations for the diagnosis of cancer will not report all other findings. Thus, while the description of this pathologist report available in the record is indicative of pneumoconiosis, it is impossible for me to determine whether the extent of the Claimant's alleged pneumoconiosis was fully characterized by this pathologist.

Dr. Perper was engaged by the Claimant to review the slides made during the Claimant's biopsy and gross and microscopic examination of the resected lung lobes. Based on the slides taken from the resected upper and middle right lobes, Dr. Perper found simple coal workers' pneumoconiosis of slight and primarily macular nature, and moderately severe centrilobular emphysema. He also found "moderate, focal, dense, fibro-anthracosis with presence of clusters of numerous birefringent silica crystals" in the pleura and birefringent silica crystals in the anthracotic areas and the alveoli. On the lymph node slide, Dr. Perper found "focal anthracosis with presence of small numbers of birefringent silica crystals." On the slide from the biopsy, he found pulmonary anthracotic pigmentation. He also noted anthracotic pigmentation scattered throughout the lung parenchyma, with occasional birefringent silica crystals and "small remnants of recognizable lung tissue with interstitial fibrosis."

Dr. Perper performed an extensive evaluation of the slides made available for his review. He described the slides reviewed, noting the locations from which the samples were taken. Without the testimony of the hospital pathologist, it is impossible to ascertain with certainty that these slides are representative of the samples reviewed by the hospital pathologist, though there is no evidence in the record that these slides are unreliable, either. Furthermore, even Dr. Pickerill diagnosed "minimal" coal workers' pneumoconiosis based on the 1995 pathological findings. Whether considered as the equivalent of a qualifying biopsy report, or as part of a reasoned medical opinion, I find that Dr. Perper's opinion regarding the existence and extent of pneumoconiosis based on the slides to be persuasive. Therefore, I conclude that the biopsy is indicative of silica crystals, anthracosis and centrilobular emphysema, which Dr. Perper opined was related to coal dust exposure. I note that anthracosis is included in the regulatory definition of pneumoconiosis and that centrilobular emphysema can be considered pneumoconiosis as it is legally defined. These findings are more substantial than mere "anthracotic pigmentation," which would be insufficient evidence of pneumoconiosis as it is defined in the Act and Regulations.

I must next consider the medical opinions. The Claimant can establish that he suffers from pneumoconiosis by well-reasoned, well-documented medical reports. A “documented” opinion is one that sets forth the clinical findings, observations, facts, and other data upon which the physician based the diagnosis. *Fields v. Island Creek Coal Co.*, 10 B.L.R. 1-19, 1-22 (1987). An opinion may be adequately documented if it is based on items such as a physical examination, symptoms, and the patient's work and social histories. *Hoffman v. B&G Construction Co.*, 8 B.L.R. 1-65, 1-66 (1985); *Hess v. Clinchfield Coal Co.*, 7 B.L.R. 1-295, 1-296 (1984); *Justus v. Director, OWCP*, 6 B.L.R. 1-1127, 1-1129 (1984). A “reasoned” opinion is one in which the judge finds the underlying documentation and data adequate to support the physician's conclusions. *Fields*, above. Whether a medical report is sufficiently documented and reasoned is for the judge to decide as the finder-of-fact; an unreasoned or undocumented opinion may be given little or no weight. *Clark v. Karst-Robbins Coal Co.*, 12 B.L.R. 1-149, 1-155 (1989) (en banc). An unsupported medical conclusion is not a reasoned diagnosis. *Fuller v. Gibraltar Corp.*, 6 B.L.R. 1-1291, 1-1294 (1984). A physician's report may be rejected where the basis for the physician's opinion cannot be determined. *Cosaltar v. Mathies Coal Co.*, 6 B.L.R. 1-1182, 1-1184 (1984). An opinion may be given little weight if it is equivocal or vague. *Griffith v. Director, OWCP*, 49 F.3d 184, 186-187 (6th Cir. 1995); *Justice v. Island Creek Coal Co.*, 11 B.L.R. 1-91, 1-94 (1988); *Parsons v. Black Diamond Coal Co.*, 7 B.L.R. 1-236, 1-239 (1984).

The qualifications of the physicians are relevant in assessing the respective probative values to which their opinions are entitled. *Burns v. Director, OWCP*, 7 B.L.R. 1-597, 1-599 (1984). More weight may be accorded to the conclusions of a treating physician as he or she is more likely to be familiar with the miner's condition than a physician who examines him episodically. *Onderko v. Director, OWCP*, 14 B.L.R. 1-2, 1-6 (1989). However, a judge “is not required to accord greater weight to the opinion of a physician based solely on his status as claimant's treating physician. Rather, this is one factor which may be taken into consideration in . . . weighing . . . the medical evidence . . .” *Tedesco v. Director, OWCP*, 18 B.L.R. 1-103, 1-105 (1994). Factors to be considered in weighing evidence from treating physicians include the nature and duration of the relationship, and the frequency and extent of treatment.

In this case, every physician who examined the Claimant and/or reviewed the medical records, believed or conceded that the Claimant had pneumoconiosis. Dr. Bizousky found that the Claimant had obstructive and restrictive impairment which was partially caused by coal dust exposure. Dr. Schaaf concluded that the Claimant had pneumoconiosis, based on x-ray evidence, albeit of a low profusion. Dr. Pickerill found minimal pneumoconiosis, based on pathology. As mentioned before, Dr. Perper found pneumoconiosis, based on his review of the pathology slides. Finally, after reviewing the pathology evidence, Dr. Branscomb conceded that the Claimant may have “minimal” “microscopic” pneumoconiosis. Thus, it is clear that the medical opinions support a finding of the existence of pneumoconiosis.

While the findings of anthracosis are diagnostic of pneumoconiosis under the regulations, Dr.

Perper also opined that the centrilobular emphysema he found in the Claimant's lungs could be attributed to or made worse by exposure to coal dust. He stated that "centrilobular emphysema is a direct result of exposure to mixed coal mine containing silica and coal worker's pneumoconiosis." He also opined that coal workers' pneumoconiosis was a substantial contributing cause of the Claimant's disability "both directly and through the associated centrilobular emphysema, that caused hypoxemia that either triggered or aggravated the myocardial ischemia associated with the arteriosclerotic heart disease or aggravated the patient's ischemic heart condition following the advent of the myocardial infarction."

Dr. Pickerill found that the Claimant had chronic obstructive pulmonary disease, and in particular, found evidence of emphysema. However, he attributed this emphysema solely to cigarette smoking, noting that centrilobular emphysema is most commonly associated with smoking. The physicians of record have acknowledged that emphysema is a form of chronic obstructive pulmonary disease. Both Dr. Bizousky and Dr. Schaaf determined that the Claimant had chronic obstructive pulmonary disease, and Dr. Schaaf specifically stated that coal dust exposure can cause this kind of impairment. Dr. Branscomb, however, found that "the extent of identifiable emphysema on a microscope slide has no correlation whatsoever with the level of overall pulmonary function of the patient." He found no evidence that the Claimant had severe emphysema.

The weight of the evidence supports that the Claimant did, in fact, have emphysema. While Dr. Branscomb disputes the level of emphysema, I note that this diagnosis was mentioned in the Claimant's medical records as early as 1992. Moreover, Dr. Perper found definitive evidence of emphysema in his review of the pathology slides. Dr. Perper provided an extensive review of the literature supporting his conclusion that centrilobular emphysema may be caused, at least in part, by coal dust exposure. I certainly take into account the Claimant's extensive and unfortunate smoking history, and note that I conclude the weight of the evidence of record supports a smoking history of up to 75 pack years (1-1½ packs per day from 1945 to 1995). Nevertheless, the record also supports a history of 21 years and two months of coal mine employment. Dr. Branscomb's rebuttal to Dr. Perper's review of the literature consists of only a statement that he disputes Dr. Perper's conclusions. He does not provide any reference to countervailing studies, nor does he even discuss why Dr. Perper is incorrect. While Dr. Branscomb is very well credentialed and experienced, his conclusory statements are insufficient to overcome Dr. Perper's well reasoned and well documented finding that the Claimant's centrilobular emphysema was caused, at least in part, by coal dust exposure.

Dr. Perper also opined that lung cancer can be related to silica exposure, and he provided a review of the literature in support of that contention. Dr. Pickerill conceded that fibrosis related to silica and other dust exposure can be related to lung cancer, but opined that the type of cancer would more often be adenocarcinoma rather than squamous cell. Dr. Pickerill had seen a synergistic effect with silica exposure and cigarette smoking in causing lung cancer, but "more often when you can actually find fibrosis in the lung from the silica exposure." Dr. Bizousky did not opine that the Claimant's bronchogenic squamous cell carcinoma could be attributed to coal dust exposure. Dr. Schaaf noted

that some studies had shown a link between coal dust exposure and lung cancer, but that the link was weaker than the one between smoking and lung cancer. Dr. Schaaf could not say that the Claimant's lung cancer was caused by coal dust exposure. Dr. Branscomb did not specifically address the potential relationship between silica exposure and lung cancer. Weighing the opinion of Dr. Perper against the contrary opinion of Dr. Pickerill, and taking into account that no other physician addressed the issue of a possible relationship between silica exposure and the Claimant's lung cancer, I conclude that the weight of the evidence is insufficient to establish that the Claimant's bronchogenic squamous cell carcinoma was caused in any part by his coal mine employment.

While the x-ray evidence was not sufficient alone to find pneumoconiosis, when considered with the pathology evidence and the opinions of the physicians of record, I find that the Claimant has established the existence of pneumoconiosis.

Causal Relationship Between Pneumoconiosis and Coal Mine Employment

The Regulations provide for a rebuttable presumption that pneumoconiosis arose out of coal mine employment if a miner with pneumoconiosis was employed in the mines for ten or more years. 20 CFR § 718.203(b) (2001). The Claimant was employed as a miner for at least 21 years and two months, and therefore is entitled to the presumption. There is no countervailing evidence in the record. Therefore, I conclude that the Claimant's pneumoconiosis arose out of his coal mine employment.

Total Disability

A miner is considered totally disabled if he has complicated pneumoconiosis, 20 CFR § 718.304 (2001), or if he has a pulmonary or respiratory impairment to which pneumoconiosis is a substantially contributing cause, and which prevents him from doing his usual coal mine employment and comparable gainful employment, 20 CFR § 718.204(b) and (c) (2001). The Regulations provide five methods to show total disability other than by the presence of complicated pneumoconiosis: (1) pulmonary function studies; (2) blood gas studies; (3) evidence of cor pulmonale; (4) reasoned medical opinion; and (5) lay testimony. 20 CFR § 718.204(b) and (d) (2001). Lay testimony may only be used in establishing total disability in cases involving deceased miners, and in a living miner's claim, a finding of total disability due to pneumoconiosis cannot be made solely on the miner's statements or testimony. 20 CFR § 718.204(d) (2001); *Tedesco v. Director, OWCP*, 18 B.L.R. 1-103, 1-106 (1994). There is no evidence in the record that Mr. Semsick suffers from complicated pneumoconiosis or cor pulmonale. Thus I will consider pulmonary function studies, blood gas studies and medical opinions.

There are four pulmonary function tests of record. There is one from 1992, the results of which are not qualifying. There are two from 1999. The September 1, 1999, pulmonary function test does not have qualifying values. The November 18, 1999, pulmonary function test does have qualifying values, both pre and post bronchodilator. The February 15, 2000, pulmonary function test is not

qualifying. I accord less weight to the 1992 test, as the Claimant's pulmonary condition has clearly deteriorated since that time, due to his bronchogenic squamous cell carcinoma and resulting resection of the right upper and middle lobes of his lungs. Nevertheless, of the three more recent pulmonary function tests, only one has qualifying values. Without reaching the issues of reliability and validity, the Claimant clearly cannot establish by weight of the pulmonary function test evidence that he has a totally disabling impairment. However, I will revisit the issue of the pulmonary function tests in the context of the medical opinion evidence, as the pattern of impairment, though not qualifying, lends credence to the medical opinions regarding the Claimant's level of impairment.

Turning next to the arterial blood gas studies, neither of the two studies were qualifying. Most of the physicians of record characterized the Claimant's arterial blood gas studies as "normal." Obviously the Claimant cannot rely on the arterial blood gas study evidence to establish total disability.

Therefore, I now consider whether the medical opinion evidence establishes that the Claimant has a totally disabling pulmonary impairment. Dr. Bizousky felt that Claimant had a severe pulmonary impairment. Dr. Schaaf felt the Claimant had severe obstructive airways disease, and that he could not return to his last coal mine employment due to multiple factors. Based on the Claimant's FEV1/FVC ratio, he considered the Claimant's level of obstruction to be severe. Dr. Pickerill felt that Claimant had a moderate functional impairment and that he was disabled due to chronic obstructive pulmonary disease, in addition to the Claimant's lung resection and other diseases of a non-pulmonary nature. Dr. Perper agreed with other physicians that the Claimant was disabled due to a number of conditions including pulmonary problems and non-pulmonary conditions. Dr. Branscomb felt that the Claimant's pulmonary condition contributed to his total disability.

The Claimant's non-pulmonary, non-respiratory impairments are relevant only to the extent that they caused a pulmonary or respiratory impairment. In this case, the physicians have characterized the Claimant's level of obstructive impairment at various levels from mild to moderate, to severe in nature. In addition, none of the physicians appear to believe that the Claimant is capable of returning to his last coal mine employment, though this finding is based on the Claimant's multiple other medical problems.

I find that the greater weight of the evidence suggests that the Claimant is totally disabled from a purely pulmonary/respiratory standpoint. While he certainly suffers from a variety of other non-pulmonary, non-respiratory impairments that are also totally disabling in nature, this does not discount the severity of his pulmonary/respiratory impairment.

The Claimant underwent a lung resection as treatment for his bronchogenic squamous cell cancer in 1995, which involved removal of one-half to two-thirds of his right lung. It was noted that the Claimant's FEV1 and FVC would be proportionally reduced, such that the Claimant's FEV1/FVC ratio (the measure of obstruction) would not be very affected by the lung resection. In 1999, the Claimant's pre-bronchodilator FEV1/FVC ratio was as low as 47%. Dr. Schaaf characterized this impairment as "severe." However, on the whole, the Claimant's level of obstructive impairment was the

same before and after his lung resection surgery. This indicates that the obstructive impairment was not due solely to the lung cancer. Nevertheless, there was a marked reduction in both his FEV1 and FVC between 1992 and 1999. The latter pulmonary function tests, while not qualifying, were very close to meeting the regulatory requirements for qualifying values. Based on the medical reports finding moderate to severe obstruction and the Claimant's lung resection, I conclude that the Claimant is totally disabled from a pulmonary/respiratory standpoint. In making this determination, I have given due regard to the Claimant's myriad other health problems. I note that Dr. Schaaf concluded that the Claimant's breathlessness could not be attributed to his cardiac disease, since the Claimant was not in heart failure. While I have no doubt that the Claimant's cardiovascular problems are also totally disabling, I specifically conclude that discounting the Claimant's cardiovascular problems, age, and amputation, he would still be totally disabled based solely on his pulmonary and respiratory difficulties. Some of the reviewing and treating physicians expressed opinions that were less than clear regarding the etiology of the Claimant's total disability, in that they noted the many problems which would prevent the Claimant from returning to his last coal mine employment. However, there is substantial evidence that every physician, with the possible exception of Dr. Branscomb, believed the Claimant would be incapable of returning to his last coal mine employment from a pulmonary standpoint alone.

Causation of Total Disability

In order to be entitled to benefits, the Claimant must establish that pneumoconiosis is a "substantially contributing cause" to his disability. A "substantially contributing cause" is one which has a material adverse effect on the miner's respiratory or pulmonary condition, or one which materially worsens another respiratory or pulmonary impairment unrelated to coal mine employment. 20 CFR § 718.204(c) (2001); *Bonessa v. U.S. Steel Corp.*, 884 F.2d 726, 734 (3rd Cir. 1989). Mere minimal contribution does not satisfy this standard.

I have concluded that the Claimant's centrilobular emphysema and anthracosis were caused at least in part by exposure to coal mine dust. Therefore, I must decide whether these conditions were a substantial contributor to the Claimant's total pulmonary disability. I find that they are. While Dr. Pickerill and Dr. Branscomb may be correct that the Claimant's anthracosis is minimal in nature, I do not find persuasive Dr. Branscomb's conclusion that the Claimant's emphysema was not severe. Indeed, most physicians of record noted that the Claimant had a moderate to severe obstructive defect. Dr. Schaaf explained that this defect could not be accounted for by the lung resection in and of itself, because the drop in FEV1 and FVC would be proportional, therefore not significantly disturbing the FEV1/FVC ratio. Accordingly, I conclude that the Claimant's coal workers' pneumoconiosis, as it is legally defined, was a substantial contributor to his total pulmonary disability.

FINDINGS AND CONCLUSIONS REGARDING ENTITLEMENT TO BENEFITS

The Claimant has met his burden to establish that he is totally disabled due to pneumoconiosis and is therefore entitled to benefits under the Act.

ATTORNEY'S FEES

The Regulations address attorney's fees at 20 CFR §§ 725.362, 365 and 366 (2001). Claimant's attorney has not yet filed an application for attorney's fees. Claimant's attorney is hereby allowed thirty days (30) days to file an application for fees. A service sheet showing that service has been made upon all parties, including the Claimant, must accompany the application. The parties have ten days following service of the application within which to file any objections. The Act prohibits the charging of a fee in the absence of an approved application.

ORDER

The claim for benefits filed by Walter Semsick, Jr., on August 9, 1999, is hereby GRANTED.

A
Alice M. Craft
Administrative Law Judge

NOTICE OF APPEAL RIGHTS: Pursuant to 20 CFR § 725.481 (2001), any party dissatisfied with this decision and order may appeal it to the Benefits Review Board within 30 days from the date of this decision and order, by filing a notice of appeal with the Benefits Review Board at P.O. Box 37601, Washington, DC 20013-7601. A copy of a notice of appeal must also be served on Donald S. Shire, Esq. Associate Solicitor for Black Lung Benefits. His address is Frances Perkins Building, Room N-2117, 200 Constitution Ave., NW, Washington, D.C. 20210.